L7 ANSWER 1 OF 2 MEDLINE on STN
ACCESSION NUMBER: 2000029768 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 10562283
TITLE: Comparative analysis of p73 and p53 regulation and effector functions.
AUTHOR: Fang L; Lee S W; Aaronson S A
CORPORATE SOURCE: Derald H. Ruttenberg Cancer Center, Mount Sinai School of Medicine, New York, New York 10029, USA.
CONTRACT NUMBER: CA66654 (United States NCI)
CA78356 (United States NCI)
CA82211 (United States NCI)
SOURCE: The Journal of cell biology, (1999 Nov 15) Vol. 147, No. 4, pp. 823-30.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199912
ENTRY DATE: Entered STN: 13 Jan 2000
Last Updated on STN: 13 Jan 2000
Entered Medline: 17 Dec 1999

AB p53 is mutated in approximately 50% of human cancers, whereas mutations of the related p73 gene are rare. p73 can activate p53-responsive promoters and induce apoptosis when overexpressed in certain p53-deficient tumor cells. We show that p73 isoforms, p73alpha and p73beta, can each induce permanent growth arrest with markers of replicative senescence when overexpressed in a tetracycline-regulatable manner in human cancer cells lacking functional p53. Human homologue of mouse double minute 2 gene product (hMDM2), but not an NH(2)-terminal deletion mutant, coimmunoprecipitated with p73alpha or p73beta, and inhibited p73 transcriptional activity as with p53. In contrast to p53, ectopically expressed hemagglutinin (HA)-tagged p73 proteins were not stabilized by treatment with several DNA damaging agents. Furthermore, unlike normal p53, which increases in response to DNA damage due to enhanced protein stability in MCF7 cells, endogenous p73 protein levels were not increased in these cells under the same conditions. Thus, although p73 has an ability, comparable to that of p53, to suppress tumor cell growth in p53-deficient cells, p73 induction is regulated differently from p53. These findings suggest that the selective pressures for p53 rather than p73 inactivation in tumors may reflect their differential responses to stresses such as DNA damage, rather than their capacities to induce permanent growth arrest or apoptosis programs.

L7 ANSWER 2 OF 2 MEDLINE on STN
ACCESSION NUMBER: 1995380270 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 7651818
TITLE: A functional p53-responsive intronic promoter is contained within the human mdm2 gene.
AUTHOR: Zauberman A; Flusberg D; Haupt Y; Barak Y; Oren M
CORPORATE SOURCE: Department of Chemical Immunology, Weizmann Institute of Science, Rehovot, Israel.
CONTRACT NUMBER: R01 CA40099 (United States NCI)
SOURCE: Nucleic acids research, (1995 Jul 25) Vol. 23, No. 14, pp. 2584-92.
Journal code: 0411011. ISSN: 0305-1048.
PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U28935
ENTRY MONTH: 199509
ENTRY DATE: Entered STN: 5 Oct 1995
Last Updated on STN: 3 Feb 1997
Entered Medline: 27 Sep 1995

AB The mdm2 gene is a target for transcriptional activation by the p53 tumor suppressor gene product. Previous work has revealed that the mouse mdm2 gene contains two promoters: one is located upstream to the gene and is active in the absence of p53, the other resides within the first intron and requires p53 for transcriptional activity. To determine whether this unique promoter activation pattern is biologically important, we investigated the structure and function of the corresponding region of the human mdm2 (hmdm2) gene. We report here that the hmdm2 gene also contains an intronic, p53-dependent promoter. The structural features of this promoter are highly conserved between mouse and man, as opposed to the lack of conservation of the first exon. This promoter is triggered in vivo in the presence of activated wild type p53, leading to the production of novel mRNA species. The intronic hmdm2 promoter contains two tandem p53 binding elements. Deletion analysis suggests that optimal promoter activity requires the simultaneous binding of p53 to both elements; this may serve to prevent premature triggering of the promoter by p53.

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(FILE 'HOME' ENTERED AT 15:19:39 ON 18 DEC 2008)

FILE 'MEDLINE, HCAPLUS, EMBASE, BIOTECHDS' ENTERED AT 15:20:15 ON 18 DEC 2008

L1 0 S HUMAN MOUSE DOUBLE MINUTE 2 HOMOLOG
L2 12 S MINUTE 2 HOMOLOG
L3 8 DUP REM L2 (4 DUPLICATES REMOVED)
L4 36 S (HUMDM2 OR HMDM2)
L5 18 DUP REM L4 (18 DUPLICATES REMOVED)
L6 15 S L5 AND (CDNA OR DNA OR GENE)
L7 2 S L5 AND (CDNA OR DNA OR GENE) AND HOMOLOG?